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CIGUATERA FISH POISONING LINKED TO THE INGESTION OF BARRACUDA IN A MONTREAL RESTAURANT — QUEBEC

Ciguatera fish poisoning in humans is most commonly caused by eating carnivorous fish contaminated by a toxin produced by the dinoflagellate *Gambierdiscus toxicus*. The toxin bio-accumulates through the food chain, and carnivorous fish such as barracuda, red snapper, grouper, sturgeon, and amberjack are usually the most commonly involved.

Most cases of ciguatera poisoning reported in the past have involved Canadians who ate fish in the Caribbean while on holiday. These cases have implicated grouper in the Dominican Republic^(1,2), kingfish in Jamaica, and barracuda in Haiti⁽³⁾. A fish casserole caused the illness in 57 people during a trip to Cuba⁽⁴⁾. Ciguatera poisonings have also been reported in Canada as a result of importing certain fish. Dried barracuda brought back from Jamaica in 1983 and imported red snapper bought in an Ontario market were identified sources⁽⁵⁾. This report describes an incident of ciguatera poisoning which occurred among five patrons of a Montreal restaurant. This appears to be the first outbreak of ciguatera poisoning in Quebec resulting from eating fish in a restaurant.

On 11 November 1996, the menu of a Montreal restaurant offered oven-baked barracuda, served with vegetables and stewed tomatoes. In all cases, the fish was prepared in the same manner and eaten at lunchtime. All five cases became ill the same day they ate the meal. On 21 November 1996, a medical doctor advised the Infectious Disease Unit (IDU) of the *Direction de la santé publique de Montréal-Centre* of two cases of ciguatera fish poisoning in persons who had eaten barracuda in the Montreal restaurant.

Case 1

A 34-year-old female ate the barracuda at approximately 12:30 hours. At approximately 15:00 hours, she experienced vomiting and diarrhea. The following neurologic symptoms presented themselves concurrently: paresthesia in the form of numbness in

the hands and feet, pruritus, and sensitivity to air, which was described as a burning sensation on the skin following exposure to the surrounding air. She also felt that she was lightheaded and going to lose consciousness, and that her thought processes had slowed down (e.g. forgetting words, names of objects, and inverted words). She experienced shivering the following day and arthralgia in the knee 2 days later. On 12 November, she experienced temperature reversal. Her pruritus symptoms were treated with diphenhydramine chlorhydrate (Benadryl[®]) which mitigated the itching.

Case 2

A 65-year-old male ate the barracuda around noon. His symptoms, which initially were diarrhea and shivering, began to appear around 17:30 hours. He treated himself with loperamide (Imodium[®]) and 24 hours later his symptoms eased. After 4 days, the patient showed signs of pruritus mainly on the hands and feet, and temperature reversal. A rash also appeared on his hands, feet, and genitalia. He noted that his symptoms had increased after eating an evening meal with his usual glass of wine. He stopped drinking the alcohol and his pruritus cleared up considerably. He described his sense of touch at the tips of his fingers and the soles of his feet as a constant silky sensation.

Case 3

A 41-year-old female ate the barracuda around 13:15 hours. Her symptoms appeared at approximately 15:30 hours and consisted mainly of epigastric burning, copious vomiting, diarrhea, abdominal pain, shivering, and dyspnea. That evening, she consulted a doctor in a clinic where her blood pressure dropped and she lost consciousness. She was not hospitalized. On 12 November, she reported pruritus on her hands, feet, and head, as well as temperature reversal. She felt as if the water she drank was sparkling. Other reported symptoms included nausea, weakness, muscular spasms, and lightheadedness.

Case 4

A 31-year-old woman ate the barracuda at lunch with Case 5 at approximately 14:15 hours. Around 17:15 hours she experienced vomiting, diarrhea, cephalalgia, and felt lightheaded. On 12 November, she experienced a feeling of paresthesia of the fingers and feet, and temperature reversal. Moreover, when her hands, mouth, or tongue came into contact with a cold object or cold water, she had a prickly sensation. She experienced pruritus, muscular cramps in the lower limbs, and a feeling of heaviness in both lower and upper limbs. Other symptoms were nausea and fatigue.

Case 5

A 44-year-old male who ate lunch with Case 4 at approximately 14:15 hours felt the onset of symptoms about 17:30 hours. The initial symptom was diarrhea, followed by nausea and shivering around 20:00 hours. He experienced temperature reversal and pruritus on his hands and feet that night. He was unable to remain standing, and he experienced a serious episode of diaphoresis together with lightheadedness. An ambulance was called; his pulse varied between 35 and 40 beats per minute upon its arrival. He was given oxygen and intravenous medication, and transferred to hospital where he was kept for observation for 48 hours to eliminate any possibility of pathologic cardiac ischemia. The results of a stress test done following his discharge from hospital were negative. However, he experienced double vision, a definite weakness in the lower limbs, dental pain, facial pain, dysphasia, and lightheadedness.

Summary

The average age of the persons affected was 40.3 years (range: 31 to 65). The initial symptoms were gastrointestinal in 100% of the cases and neurologic in 20%. One single case presented both types of symptoms simultaneously. The average incubation period was 3 hours and 5 minutes (range: 1 hour and 45 minutes to 5 hours). Five individuals ate the barracuda at this restaurant and all five became sick, i.e. 100% were affected. Four cases were contacted 6 months later to verify if they still had any symptoms. Each had experienced temperature inversion which had disappeared within 2 to 4 months. Case 1 reported intermittent spontaneous burning sensations upon contact with the surrounding air as well as pruritus. Case 2 had no symptoms after the initial attack. Case 3 reported myalgia at the time of contact. Case 4 reported pruritus after consuming alcohol and certain foods (e.g. cheese and certain types of meat). This case also mentioned insomnia, dizziness, and aching in the jaw.

Food Inquiry

A patron contacted the restaurant during the afternoon of 11 November and reported having experienced gastrointestinal symptoms a few hours after eating the barracuda. The manager immediately ordered that all of the fish be thrown out. An inspector from the food inspection division of the *Communauté urbaine de Montréal*, who had been notified by the IDU, arrived the morning of 22 November to undertake an inspection but there was no barracuda left for analysis. A verification of the invoices confirmed that the barracuda had been purchased from a Montreal wholesaler. The *Ministère de l'Agriculture, des Pêcheries et de l'Alimentation* took over the investigation and contacted the supplier.

The restaurant manager had purchased all of the barracuda – about 5 kg that was available from a Montreal wholesaler. The

wholesaler had obtained his supply from a second Montreal wholesaler with headquarters in Ontario. The fish had originally been purchased from an American wholesaler in Georgia on 31 October as part of a large lot of various types of fish. The total amount of barracuda in this lot was 216 kg with 5 kg going to Montreal and the remaining quantity destined for sale as fresh fish in Ontario. The imported fish had been cut up, and it was impossible to trace the pieces that had been sold in Ontario; moreover, the details of the importing process could not be followed precisely. However, according to various wholesalers who were interviewed, workers in the fish industry are informed of the risks of ciguatera poisoning associated with certain types of tropical fish. Furthermore, it is recognized that these types of fish should be imported from non-coral areas because *Gambierdiscus toxicus* grows on coral reefs.

Discussion

The symptoms experienced by the cases in this incident are typical of ciguatera fish poisoning⁽⁶⁾. Ciguatoxin is thermostable, and is not destroyed by cooking or other fish-processing methods. It is odourless and does not alter the taste or appearance of the fish. Therefore, it is impossible to easily identify a fish that may be contaminated by the toxin.

This type of poisoning may be prevented by informing consumers of the potential risks of ciguatera poisoning associated with certain types of fish; highly susceptible species should be avoided. If one wishes to eat them, it is extremely important to avoid the larger varieties such as barracuda. As a general rule, large mature carnivorous fish, which are located at or near the top of the food chain, are more susceptible to ciguatoxin contamination, and the toxin is likely to be present in higher amounts in these fish.

At the present time in Canada, there are no regulations restricting the importation of barracuda⁽⁷⁾. The federal government quality management program for importers is currently investigating ciguatera toxin levels in imported tropical fish; lots of contaminated fish will be refused entry into Canada (Y. Roy, Canadian Food Inspection Agency, Montreal: personal communication, 1997).

Conclusion

Ciguatera fish poisoning is usually detected among returning travellers. However, it is possible to be exposed to ciguatoxin and to experience its symptoms without leaving Canada. When presented with a pathognomonic symptomatology involving temperature reversal, a diagnosis of possible ciguatera poisoning should be considered, even if there is no prior history of travelling.

Acknowledgements

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Source: A Bruneau, MD, Direction de la santé publique de Montréal Centre, S Mahant, MD, T Al-Azraqi, MD, J MacLean, MD, Montreal General Hospital, M Bourque, DVM, Ministère de l'Agriculture, des Pêcheries et de l'Alimentation, F Desroches, Communauté urbaine de Montréal, Montreal, QC.

International Notes

TRANSMISSION OF HEPATITIS C VIRUS INFECTION ASSOCIATED WITH HOME INFUSION THERAPY FOR HEMOPHILIA

Transmission of hepatitis C virus (HCV) and other bloodborne viruses between household members who are not sex partners presumably results from inapparent percutaneous or perimucosal exposures, such as sharing articles that may be contaminated with microscopic quantities of blood. The risk for nonsexual household transmission is extremely low, and no cases of such transmission have been documented⁽¹⁾; direct percutaneous exposures (e.g., injecting drugs) have been identified as the major risk factor for infection⁽¹⁾. This report summarizes the investigation of a newly acquired case of HCV infection in a child with hemophilia, after a preliminary investigation identified several household members with HCV infection. The findings suggest the child acquired infection through percutaneous exposure to the mother's HCV-infected blood during infusion of clotting-factor concentrate.

On 12 September, 1996, a case of seroconversion of antibody to HCV (anti-HCV) in a 4-year-old child with moderate factor VIII deficiency was reported to the Seroconversion Surveillance Project, a surveillance system maintained jointly by the Food and Drug Administration, Centers for Disease Control and Prevention (CDC), and the National Hemophilia Foundation. The child tested positive for anti-HCV on 29 August 1996, after testing negative in June 1994 and August 1995. Serum drawn on the same day (29 August) tested negative for human immunodeficiency virus (HIV) antibody. With the exception of the 14 days after birth, the child had always received recombinant clotting-factor concentrate for treatment of bleeding episodes.

Testing of serum samples from six household members indicated that three were anti-HCV-positive, including the patient's mother, an older sibling, and an aunt who had stayed in the household for 6 weeks during September and October 1995. The mother and aunt had histories of having injected illicit drugs but had not been tested previously for anti-HCV. The sibling, aged 11 years, had moderate factor VIII deficiency and was anti-HCV-positive when first tested in 1992.

Until November 1994, the child was treated for bleeding episodes at a local emergency department with recombinant clotting-factor concentrate brought from home. Beginning in November 1994, the patient's mother administered clotting-factor concentrate to him at home after receiving training from a nurse employed by a home health-care company. Follow-up consisted of an annual visit to a hemophilia treatment centre. During February 1995 to June 1996, the period during which the child probably became infected, the patient's mother administered factor VIII concentrate to him on 13 occasions. She reported that, until May

1996, three other persons were required to restrain the child during infusions because the child was combative and resistant. Infusions usually were administered through a vein in the foot because of reported difficulties in accessing a vein in the upper arm, and up to 3 hours were required for the infusion. The mother recalled that, on at least two occasions, she pricked her finger with the needle while attempting an infusion and drew a visible quantity of blood, but she could not remember whether she continued to use the same needle for the infusion. Before learning in September 1996 that she was infected with HCV, she did not use gloves when infusing clotting-factor concentrate. No other family members assisted in administering factor concentrates.

The child and mother shared a bed. Although each household member had his or her own toothbrush, bath towels were shared. All household members were negative for or denied recent histories of dermatitis, open wounds, injury, or external bleeding episodes.

Sequence analysis of the HCV strains of the child and the HCV-infected family members indicated that the strain isolated from the mother and the child were identical in sequence of 220 nucleotides in the NS5b region of the genome. Viral sequences in this region isolated from the aunt and brother differed by four and 10 nucleotides, respectively, from the child's strain.

MMWR Editorial Note

The results of the investigation described in this report suggest that the child acquired HCV infection through percutaneous exposure to the mother's HCV-infected blood during infusion of clotting-factor concentrate. The mother was responsible for infusing factor concentrate and reported incurring needlesticks during some of these infusions. Therefore, blood-to-blood contact may have resulted either from use of a contaminated needle to administer an infusion or by contamination of the infusion site. In addition, analysis of the sequences of the segments of HCV strains isolated from the mother and child indicated the strains were closely related. Because the time of initial infection of the mother could not be documented, the possibility that the child acquired infection from another unrecognized source and was the subsequent source of infection for the mother cannot be excluded. However, the mother had been a long-term injecting-drug user before the birth of the child and may have acquired HCV infection through sharing needles and syringes. Surveys indicate that up to 90% of long-term injecting-drug users test positive for anti-HCV⁽¹⁾.

Among persons with hemophilia who were heavily infused with clotting-factor concentrates before the development of viral inactivation methods, the prevalence of anti-HCV exceeds 90%⁽¹⁾. The safety of plasma-derived clotting-factor concentrates has been improved by instituting measures that include screening for serologic markers of bloodborne pathogens in donated plasma used in the manufacture of these products and the incorporation of viral inactivation steps (e.g. dry heating, pasteurization, and solvent detergent treatment)⁽²⁾. Transmission of HCV or other viral agents has not been reported in association with receipt of genetically engineered factor concentrates or of albumin, the only human plasma-derived material present in these recombinant products^(3,4). Based on these considerations, clotting-factor concentrate was an unlikely source of infection in the case described in this report because the child had received only recombinant product during the period in which infection was likely to have been acquired.

Home infusion therapy is a convenient and cost-effective alternative to treatment of hemophilia in the health-care setting⁽⁵⁾. However, if proper infection-control procedures are not followed, patients and household members may be at risk for exposure to bloodborne pathogens during home infusion therapy. In one study, 18% of household members who assisted HIV-infected hemophilia patients with the infusion process reported having sustained at least one needlestick injury⁽⁶⁾, and HIV infection has been acquired through percutaneous exposure during home treatment of acquired immunodeficiency syndrome⁽⁷⁾ and hemophilia⁽⁸⁾.

CDC recommends that patients and families who are eligible for home infusion therapy be informed of the potential risks for infection with bloodborne pathogens and be assessed for their ability to use adequate infection-control practices consistently. Patients and families should receive training with a standardized curriculum that includes appropriate infection-control procedures before initiation of home infusion therapy, and infection-control practices should be regularly evaluated at home through follow-up visits by health-care professionals with specific training in such practices. Routine testing of caregivers for bloodborne pathogens is not recommended; all caregivers should follow the universal precautions recommended for all persons who infuse blood products. Gloves should be worn by persons who prepare or infuse

blood products and during disposal of infusion equipment and waste. A needle that has broken the skin should not be reused, and used needles should never be recapped. Used needles should be placed in a sharps container in a location inaccessible to children. Needlestick incidents occurring during home infusion therapy should be reported to the health-care professionals supervising home treatment. All household and sexual contacts of patients with chronic hepatitis B virus infection should receive hepatitis B vaccine.

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Announcement

THE FOURTH INTERNATIONAL CONFERENCE ON HEMORRHAGIC FEVER WITH RENAL SYNDROME AND HANTAVIRUSES

Atlanta, Georgia, USA
5 to 7 March 1998

This conference, hosted by the Centers for Disease Control and Prevention (CDC) and co-sponsors, will facilitate the exchange of scientific information in the following areas: 1) Clinical Aspects, 2) Laboratory Diagnostics, 3) Pathogenesis and Immune Response, 4) Hantavirus Ecology, 5) Hantavirus Epidemiology, 6) Molecular Biology and Cell Interactions, 7) Health Education and Prevention, and 8) Antiviral and Vaccine Development. The meeting will include plenary sessions with invited speakers as well as oral and poster sessions based on accepted abstracts on the topics listed above.

For further information, contact Amy Corneli, Special Pathogens Branch, CDC, 1600 Clifton Road, M/S A26, Atlanta, Georgia, 30333; telephone 404-639-1510; FAX 404-639-1509; <http://www.cdc.gov/ncidod/diseases/hanta/hantconf.htm>, e-mail akc8@cdc.gov

This conference will precede the International Conference on Emerging Infectious Diseases to be held 8 to 12 March 1998 in Atlanta.